Comparative Endocranial Vascular Changes Due to Craniosynostosis and Artificial Cranial Deformation

VALERIE DEAN O'LOUGHLIN
Medical Sciences Program, Indiana University, Bloomington, Indiana 47405

KEY WORDS Endocast, Venous sinuses, Middle meningeal vessels, Cranial deformation, Craniosynostosis, Endocranium

ABSTRACT The processes of craniosynostosis (premature fusion of one or more of the calvarial sutures) and artificial cranial deformation are similar since both can alter the shape of the craniofacial complex. Most research exploring these processes has focused on the ectocranium, although it is obvious that these processes also modify the endocranium. Endocranial changes due to either craniosynostosis or artificial cranial deformation have not been as thoroughly examined.

Silicone rubber endocasts were made from 11 craniosynostotic archaeologically derived specimens from North and South America. For comparative purposes, endocasts were made from 22 normal and 17 occipitally deformed crania that were archaeologically derived from North and South America. With all samples, middle meningeal vessel patterns and venous sinus impressions were qualitatively and quantitatively analyzed. Depth, width, and convolution of the middle meningeal vessels were recorded, and the direction of vessel branches was noted.

Both artificial cranial deformation and craniosynostosis altered the endocranial vasculature. Middle meningeal vessel and venous sinus impressions of the craniosynostotic group differed when compared to both the undeformed and artificially cranially deformed samples. Sinuses traversing under synostosed sutures became wider and deeper. In contrast, sinuses directly underneath the greatest artificial deformational stress were shallower, while there was compensatory enlargement of sinuses further away from the greatest deformational effects. Such compensatory enlargement also was shown by the high incidence of enlarged occipital/marginal sinuses in artificially deformed skulls. Increased intracranial pressure is hypothesized to be the cause of the venous sinus changes found in craniosynostotic individuals. Middle meningeal vessel patterns from craniosynostotic and artificially deformed specimens were similar in that their direction paralleled the direction of altered cranial growth. These findings demonstrate that the endocranial vasculature is developmentally plastic and responds to deformation in a predictable pattern.

The craniofacial complex is developmentally plastic, both ectocranially and endocranially. This plasticity can be understood by examining the growth processes of the skull under the influence of constraints, such as those brought about by craniosynostosis or artificial cranial deformation. Moss's "functional craniology" (Moss and Young, 1960) states that the growth of the endocranial components (which includes the brain, meninges, and blood vessels) is responsible for the passive outward movement of the

Received June 21, 1994; accepted May 21, 1996.

Articles published before May 1996 are under the author's maiden name, Valerie L. Dean.

calvarial bones. Both artificial cranial deformation (Moss, 1958) and craniosynostosis (Moss, 1959) redirect endocranial growth, thereby altering ectocranial shape. Although Moss (1958, 1959) alludes to changes in the brain and blood vessels, he does not directly examine them. If indeed the endocranial elements are directly responsible for ectocranial shape, then their responses under different deformational forces must be examined. Such deformational forces that modify skull shape include craniosynostosis and artificial cranial deformation.

Craniosynostosis is the premature fusion of one or more of the calvarial sutures (Cohen, 1986a; Bixler and Ward, 1987). Craniosynostosis may be isolated (no other abnormalities are present except those directly caused by early suture closure), or it may be syndromic (one of many morphological manifestations of syndromes such as Apert's and Crouzon's syndromes) (Cohen, 1986a). The etiology of craniosynostosis is unclear but appears to be the result of multiple factors such as genetics, biochemical abnormalities, teratogens, and fetal head constraint (Cohen, 1986b, 1988; Bixler and Ward, 1987).

The earliest description of craniosynostosis may be attributed to Vesalius from his 1543 work *De Humani Corporis Fabrica* (see Saunders and O'Malley, 1950). Virchow's nineteenth century research (see Persing et al., 1989) was followed by many (Moss, 1959; Cohen, 1986b; Delashaw et al. 1989; Persing and Jane, 1989; Bruneteau and Mulliken, 1992) who described the relationship between types of craniosynostosis and dramatic changes in craniofacial shape. Most research on craniosynostosis has been performed by clinicians and has focused primarily on classification and corrective treatment procedures (Cohen, 1986a,c; Bixler and Ward, 1987; Delashaw et al., 1989; Persing and Jane, 1989; Bruneteau and Mulliken, 1992). In contrast, anthropologists have researched craniosynostosis less extensively and often in terms of its frequency among skeletal populations (Bennett, 1967) or as an interesting anomaly in a few isolated skulls (Oetteking, 1927; Eiseley and Asling, 1944; Hohenthal and Brooks, 1960; Hauser and Kritscher, 1994). Although Virchow (see Persing et al., 1989) and a few other researchers (Eiseley and Asling, 1944; Chadduck et al., 1992) noted that craniosynostosis also produces endocranial changes in structures such as the blood vessels, subarachnoid spaces, and brain, these effects have not been as thoroughly examined.

The effects of the cultural practice of artificial cranial deformation are similar to the effects of craniosynostosis since both can affect craniofacial shape dramatically. However, the two differ in that artificial cranial deformation primarily is produced by environmental factors, whereas many craniosynostoses appear to be primarily genetically induced. Artificial cranial deformation is found worldwide (Dingwall, 1931), and its effects on the ectocranium have been extensively researched by anthropologists (Bjork and Bjork, 1964; Ossenberg, 1970; Schendel et al., 1980; Anton, 1989; Cheverud and Midkiff, 1992; Cheverud et al., 1992; Kohn et al., 1993; Holliday, 1993). In contrast, clinical studies have been few and have been concerned with the craniofacial effects of positional molding, defined as abnormal head shape due to factors such as cerebral injury, the birth process, or neonatal preference for lying on one side of the head (Bruneteau and Mulliken, 1992).

A few anthropological studies (Grupe, 1984; Dean, 1993, 1995a, 1995b) have explored the effects of artificial deformation on endocranial structures such as the brain and blood vessels. Grupe (1984) used radiographs to determine that circular deformation enlarges diploic vessels and positions them more obliquely. Analysis of a small sample of endocranial casts (Dean, 1993, 1995a) suggested that artificial cranial deformation affects both venous sinus and middle meningeal vessel patterns. Venous sinuses were narrower and shallower in the areas of greatest deformation, while there was compensatory enlargement of the vessels furthest from these areas. In addition, the direction of the middle meningeal vessel impressions paralleled the altered craniofacial growth trajectories. For example, deformed skulls with greater growth in a superior-posterior direction had middle meningeal vessels that travelled in the same direction.

The research reported here used silicone endocranial casts to explore the effects of different types of craniosynostosis on the endocranial vasculature in comparison to those seen in the endocasts of undeformed and artificially deformed skulls. Effects on the shape, depth, and direction of sinus and vessel impressions were examined. Finally, the endocranial effects of craniosynostosis and artificial cranial deformation were compared to determine similarities and differences between primarily genetically based changes in shape and environmentally based ones.

MATERIALS AND METHODS

Fifty crania from the National Museum of Natural History and the Indiana University collections representing many geographically distinct North and South American archaeological populations were analyzed. Many populations were selected because 1) wide sampling maximized possible endocranial diversity to be studied and minimizes the risk of unintentionally selecting for population-specific traits and 2) no single archaeological population examined has a sufficient number of undeformed, artificially deformed, and craniosynostotic young adult skulls. Younger individuals (ages 18–40) were selected because meningeal vessel and sinus impressions may become wider, deeper, and more prominent with advancing age (Lindblom, 1936). In addition, some craniosynostoses are not evident after age-related suture closure had already begun. Population frequencies of craniosynostosis are very low, ranging from 3–15 per 10,000 individuals (Cohen, 1986c; French et al., 1990) although these frequencies tend to be slightly higher in skeletal populations (Bennett, 1967). Thus, control for age in the craniosynostotic group was relaxed to maximize sample size. Of the 50 crania, 22 were undeformed, 9 exhibited craniosynostoses, 17 were occipitally deformed, and 2 were both artificially deformed and exhibited sagittal synostosis (see Table 1). Age was estimated by suture closure and/or dental wear, and ages ranged from 18–40 for all but two craniosynostotic skulls (whose age ranges were 40–55 years).

Craniosynostoses were determined by inspecting for premature ectocranial and endocranial fusion of the suture(s) and included sagittal synostosis, squamosal synostosis, lambdoidal synostosis, occipitomastoid synostosis, and parietomastoid synostosis. Selection favored isolated (rather than syndromic) craniosynostosis whenever possible, as any endocranial alterations in the latter group may be related to the syndrome itself rather than the direct effects of the premature suture closure. Determining isolated vs. syndromic craniosynostosis is difficult with skeletal samples, especially if the entire skeleton is not available for examination. Criteria for determining syndromic craniosynostosis, such as polydactyly, congenital heart defects, and DNA alterations (Cohen, 1986a, 1988; Bixler and Ward, 1987) cannot be observed in skulls. However, cases involving premature closure of a single suture are often isolated craniosynostoses, whereas multiple suture closure is sometimes (but not always) the result of syndromic craniosynostosis (Cohen, 1986a). Individuals with syndromic (rather than isolated) craniosynostosis also often have characteristic facial anomalies that include maxillary hypoplasia, hypertelorism, and cleft palate (Bixler and Ward, 1987, Cohen, 1988). The sample skulls with multiple premature suture closure exhibited none of these anomalies, suggesting their craniosynostoses were isolated.

The type, degree, and asymmetry of artificial cranial deformation were qualitatively determined by visual inspection. Types of deformation were those described by Neumann (1942) and Hrdlička (1910). This project focused on occipital deformation, parallelo-fronto-occipital deformation, and circular deformation. Occipital deformation is a vertical flattening of the nuchal portion of the occipital bone. Parallelo-fronto-occipital deformation flattens the frontal region and the occipital bone proper. The occipital bone is flattened obliquely, whereby the frontal and occipital bones are approximately parallel with one another. Circular deformation compresses the skull cylindrically so that the skull becomes ovoid.

Degree of deformation was classified as none, slight, moderate, or marked (Dean, 1993, 1995a, 1995b). *No cranial deformation* was defined as no evidence of craniosynostosis or artificial cranial deformation. *Slight*

deformation was some evidence of altered skull shape, but it could be overlooked in more casual observation. *Moderate deformation* was noticeable alteration of skull shape. *Marked deformation* was clear indication of drastically altered skull shape. Degree was determined by two or more visual inspections by the author and with the assistance of comparative skulls from the same geographic area.

Asymmetry was coded as none, left, or right. For example, skulls with a more flattened left occiput than right occiput were classified as having left asymmetrical occipital deformation. Asymmetry was determined by two visual inspections by the author.

Silicone rubber endocasts were prepared using methods similar to those used by Radinsky (1968) and Murrill and Wallace (1971). Endocasts were formed from one thick layer of Rhodorsil RTV 524 silicone rubber. This brand of silicone rubber vulcanizes at room temperature, thus eliminating the need to heat the endocast. The skull's external foramina were plugged with microcrystalline wax. Twenty parts silicone were mixed with one part catalyst, and this mixture was poured into the skull through the foramen magnum. The skull was continually rotated by hand until the material set (2–5 h) to insure complete coverage of all endocranial surfaces. After letting the material set in the skull overnight, the endocast was meticulously separated from all bony surfaces and extracted through the foramen magnum. The endocast was then filled with polyester batting to maintain its shape.

Features on the endocasts were examined, and comparisons were made between right and left sides (see Fig. 1). Superior sagittal, transverse, and sigmoid sinus impressions were analyzed according to their shape, direction, and depth. The methods for analysis followed those described earlier (Dean 1993, 1994, 1995a, 1995b).

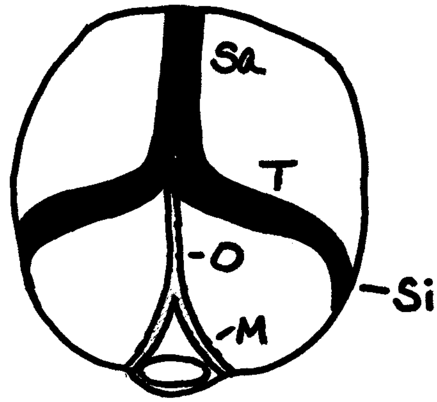
Determining vasculature impression shape can be difficult and subjective, since there is a wide range of variation in vessel width and depth. To account for these problems, the undeformed group of endocasts was visually inspected first. Each vessel was studied separately, first for width and later for depth. Undeformed endocasts were arranged in a continuum for a particular vessel width. From this continuum, endocasts were divided into four separate width categories described as small (S), medium (M), large (L), or extra large (XL). These examples were used for the other groups in establishing vessel width. Vessel depth was analyzed in a similar manner, and depth was described as shallow, medium, deep, or very deep (see Fig. 2 for comparative venous widths and depths).

Transverse and sigmoid sinus impressions also were analyzed to determine any asymmetry in venous sinus width and depth. Minor sinus impressions, such as enlarged occipital/marginal sinuses, were noted. Occipital/marginal sinuses were considered enlarged if they left a noticeable impression on the endocast. Middle meningeal vessel impressions were studied for width, depth, convolution, and orientation (Dean, 1993, 1994, 1995a, 1995b). Variation in convolution patterns was determined in a similar way to that described for vessel width and depth. Distinctions were made between the anterior (frontal) branch and posterior (parietal) branch of the middle meningeal vessels.

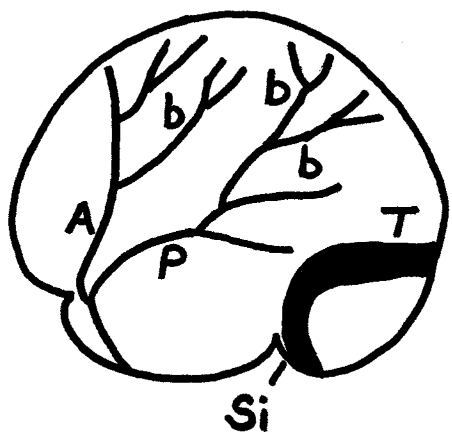
It should be noted that several smaller (and usually terminal) branches emanate off of the anterior and/or posterior branches. One of these is the middle branch of the middle meningeal vessels. The middle branch of these vessels varies as to whether it comes off of the anterior branch, the posterior branch, or both of these branches (Giuffrida-Ruggeri, 1912; Rothman, 1937; Gray, 1973). Middle meningeal vessel data were not separated according to variations in branching patterns of the middle branch because this would have produced very small group sizes. Only the anterior or posterior branch was examined when determining

Fig. 1. Illustrations and photograph of endocranial features. **A:** Posterior view: superior sagittal sinus (Sa), transverse sinuses (T), sigmoid sinuses (Si), occipital sinus (O), and marginal sinuses (M). **B:** Left view. The middle meningeal vessels are separated into an anterior branch (A) and a posterior branch (P), and each has its own smaller branches (b). **C:** Left view of an undeformed endocast, showing the features illustrated in B.

1A.



1B.



1C.

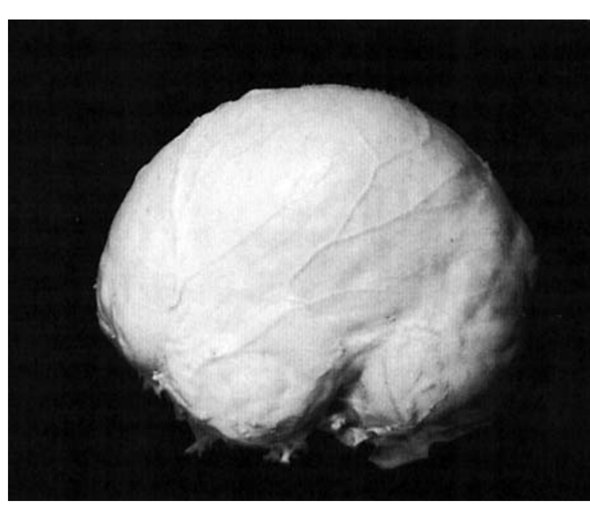


Fig. 2. Superior view of an undeformed endocast **(left)** and a sagittal synostotic endocast **(right)**. The synostotic specimen has a long, narrow cranium and an extra large, deep superior sagittal sinus impression.

width, depth, and convolution, not the middle branch or the other terminal branches. Also, determining vessel orientation was not dependent upon variations in the placement of the middle branch. Later discussion regarding directional alterations of branches refers to the terminal branches, not merely the middle branch.

Comparisons were made among craniosynostotic, artificially deformed, and undeformed endocasts on the basis of all of these observations.

RESULTS

Table 1 presents the sample profile, divided by sex and degree of deformation. The four main groups were 1) undeformed, 2) craniosynostoses, 3) occipital deformation, and 4) craniosynostoses and artificial deformation. The latter three groups were divided into subgroups (see Table 1). Sagittal synostosis is the most common type of craniosynostosis found in other research (Cohen, 1986b; Bixler and Ward, 1987), as it was for this sample. Degree of artificial deformation was either moderate or marked. Of the 17 occipitally deformed crania, 12 exhibited some form of asymmetry.

Sagittal sinus patterns

Table 2 lists the width, depth, and drainage patterns for the superior sagittal sinus. This sinus tended to drain predominantly into the right transverse sinus. The exception occurred among the six right asymmetrically occipitally deformed crania, three of which had left transverse sinus dominance.

Skulls with sagittal synostosis displayed wide, very deep sagittal sinus impressions, especially when compared to the undeformed specimens (Fig. 2). Eiseley and Asling (1944) made similar observations. The skulls that were both artificially deformed and had sagittal synostosis displayed sagittal sinus impressions resembling those seen in the undeformed and the occipitally deformed crania: medium-sized, shallow grooves.

Transverse and sigmoid sinus patterns

Tables 3 and 4 show asymmetry, width, and depth differences in the transverse and sigmoid sinuses. Right transverse sinuses tended to be wider than their left counterparts for all groups, which presumably reflects the general right-sided dominance of sagittal sinus drainage. Among the major groups, there was no significant difference in vessel depth between right and left sides. Type and asymmetry of artificial cranial deformation affected the asymmetry in width and depth between the two transverse sinuses. Right asymmetrically deformed crania tended to have wider but not necessarily deeper left transverse sinuses. Left asymmetrically deformed crania had both wider and deeper right transverse sinuses (Fig. 3). Symmetrical occipitally deformed crania displayed great diversity in transverse sinus width and depth.

Sigmoid sinuses tended to be both wider and deeper on the right side for all groups. Asymmetrical occipital deformation produced asymmetrical width and depth changes to the sigmoid sinuses.

Enlarged occipital/marginal sinuses

Table 5 lists the frequencies of enlarged occipital/marginal sinuses. Three of the four groups exhibited high frequencies of enlarged occipital and/or marginal sinuses. The size of these sinuses varied greatly, and they occasionally were larger than the transverse and sagittal sinuses (Figs. 3, 4). Occipitally deformed specimens demonstrated an especially high incidence of enlarged sinuses. Asymmetry in width and depth of these sinuses was noted for both the unde-

TABLE 1. Craniosynostosis and artificial deformation classification for sample crania

Group[1]	Sex		Artificial cranial deformation: Degree	
	M	F	Moderate	Marked
1. Undeformed	17	5		
2. Total craniosynostoses	5	4		
Sagittal synostosis only	3	2		
Squamosal synostosis only	1	1		
Sagittal + other synostoses	1	1		
3. Total occipital deformation	13	4	6	11
Left asymmetrical deformation	5	1	1	5
No asymmetry	4	1	5	0
Right asymmetrical deformation	4	2	0	6
4. Sagittal synostosis and artificial deformation	2	0	2	0
Parallelo-fronto-occipital deformation	1	0		
Circular deformation	1	0		

[1] Subgroups for the four main groups are in italics.

formed and occipitally deformed crania. In contrast, occipital/marginal sinus width and depth tended to be more symmetrical for the craniosynostotic group. Left occipitally deformed crania tended to have wider and deeper occipital/marginal sinuses on the left side (Fig. 3). Right asymmetrically deformed crania tended to have wider and deeper occipital/marginal sinuses on the right side.

Middle meningeal vessel patterns

Table 6 contains middle meningeal vessel branch width, depth, and convolution patterns for each group. As with Rothman's (1937) study of 403 male and female skulls, there were few side differences in middle meningeal vessel shape. Consequently, left and right middle meningeal vessel data were grouped together.

Table 7 summarizes the common directional and branch patterns seen in each group. The anterior and posterior branches of the middle meningeal vessels are presented separately. Side differences in asymmetrical deformation were not seen; thus, all occipitally deformed crania were grouped together for this particular analysis.

Width and depth of the middle meningeal vessels showed great diversity among the four groups (Table 6; Fig. 1, 5–8). The undeformed group showed patterns similar to that reported for the normal anatomy of these vessels (Giuffrida-Ruggeri, 1912; Rothman, 1937; Gray, 1973). However, the occipitally deformed crania had relatively wider and deeper vessels compared to the other cranial groups. The craniosynostotic group, occipitally deformed group, and craniosynostotic and artificially deformed group were similar in that all displayed more convoluted or twisted anterior branches compared to the undeformed sample.

A point of clarification is required. Both earlier studies (Wood Jones, 1912) and more recent research (Diamond, 1991, 1992; Falk and Nicholls, 1992; Falk, 1993) have suggested that the middle meningeal vessel impressions are mainly produced by the veins. Convolution in the impression is due to the impression left by the middle meningeal artery. Thus, the finding of more convoluted vessels should not be interpreted to imply that cranial deformation produces convoluted vessels. Rather, cranial deformation may merely place greater pressure on the skull, allowing for both artery and veins to leave their impression. The end result would be a more convoluted impression upon the endocast.

Directional patterns of the branches off the anterior branch varied greatly among the groups (Table 7). Sagittal synostotic individuals displayed multiple clear, long branches that slanted superior-posteriorly (Fig. 5). Here, vessel direction followed the altered increased vault length. Both craniosynostotic and deformed skulls exhibited branches that traversed in the direction of altered cranial growth (Fig. 6). Occipitally deformed specimens demonstrated an increased number of branches on the frontal lobe.

In general, the posterior branch of the

TABLE 2. Sagittal sinus patterns

Group[1]	Drainage dominance: Left transverse sinus	Drainage dominance: Equal drainage	Drainage dominance: Right transverse sinus	Width: S	Width: M	Width: L	Width: XL	Depth of vessel impression: Shallow	Depth of vessel impression: Medium	Depth of vessel impression: Deep	Depth of vessel impression: Very deep
Undeformed	3	4	15	4	15	3	0	13	7	2	0
Total craniosynostoses	0	2	7	0	6	3	0	1	3	2	3
Sagittal synostosis only	0	0	5	0	3	2	0	0	1	2	2
Squamosal synostosis only	0	1	1	0	2	0	0	0	2	0	0
Sagittal + other synotoses	0	1	1	0	1	1	0	1	0	0	1
Total occipital deformation	3	3	11	2	13	2	0	12	4	1	0
Left asymmetrical	0	1	5	2	4	0	0	5	1	0	0
No asymmetry	0	0	5	0	4	1	0	4	0	1	0
Right asymmetrical	3	2	1	0	5	1	0	3	3	0	0
Craniosynostosis and cranial deformation	0	2	0	0	2	0	0	1	1	0	0

[1]Subgroups are in italics.

TABLE 3. Transverse sinus patterns

Group[1]	Asymmetry: Larger transverse sinus?: Left	Equal	Right	Left Width: S	M	L	XL	Left Depth: Shallow	Medium	Deep	Very deep	Right Width: S	M	L	XL	Right Depth: Shallow	Medium	Deep	Very deep
Undeformed	3	6	13	5	14	3	0	9	11	2	0	2	15	4	1	9	13	0	0
Total craniosynostoses	0	5	4	5	3	1	0	6	1	1	1	0	8	1	0	7	1	0	1
Sagittal synostosis only	0	2	3	3	2	0	0	4	0	1	0	0	5	0	0	5	0	0	0
Squamosal synostosis only	0	1	1	1	1	0	0	1	1	0	0	0	2	0	0	1	1	0	0
Sagittal + other synostoses	0	2	0	1	0	1	0	1	0	0	1	0	1	1	0	1	0	0	1
Total occipital deformation	4	3	10	7	6	4	0	6	10	1	0	2	7	6	2	4	10	3	0
Left asymmetrical	1	0	5	5	1	0	0	2	4	0	0	1	3	1	1	0	4	2	0
No asymmetry	0	1	4	2	3	0	0	2	3	0	0	0	1	3	1	2	2	1	0
Right asymmetrical	3	2	1	0	2	4	0	2	3	1	0	1	3	2	0	2	4	0	0
Craniosynostosis and cranial deformation	0	1	1	1	1	0	0	1	0	0	1	0	2	0	0	1	0	0	1

[1]Subgroups are in italics.

TABLE 4. Sigmoid sinus patterns

Group[1]	Asymmetry: Larger sigmoid sinus?			Left: Width				Left: Depth				Right: Width				Right: Depth			
	Left	Equal	Right	S	M	L	XL	Shallow	Medium	Deep	Very deep	S	M	L	XL	Shallow	Medium	Deep	Very deep
Undeformed	6	5	11	8	8	5	1	7	11	5	0	3	9	10	0	3	15	4	0
Total craniosynostoses	1	3	5	5	3	1	0	1	7	1	0	1	4	4	0	1	2	5	1
Sagittal synostosis only	0	1	4	3	2	0	0	0	4	1	0	0	2	3	0	0	1	4	0
Squamosal synostosis only	1	0	1	1	0	1	0	0	2	0	0	0	1	1	0	0	1	0	1
Sagittal + other synostoses	0	2	0	1	1	0	0	1	1	0	0	1	1	0	0	1	0	1	0
Total occipital deformation	2	4	11	8	5	3	1	3	14	0	1	0	8	4	5	3	12	1	1
Left asymmetrical	0	1	5	5	1	0	0	1	5	0	0	0	2	1	3	1	3	1	1
No asymmetry	0	0	5	2	3	0	0	2	3	0	0	0	2	1	2	1	4	0	0
Right asymmetrical	2	3	1	1	1	3	1	0	6	0	1	0	4	2	0	1	5	0	0
Craniosynostosis and cranial deformation	0	1	1	1	1	0	0	1	0	1	0	0	1	1	0	0	1	1	0

[1]Subgroups are in italics.

Fig. 3. Posterior view of a left asymmetrical occipitally deformed endocast. Note the wider, deeper right transverse sinus and the enlarged bilateral occipital/marginal sinus pattern.

middle meningeal vessels was relatively straight and of medium width and depth (Table 6). However, directional pathways of these vessels showed great variety (Table 7). The craniosynostotic sample had very long straight vessels that coursed posteriorly, again reflecting the effect of increased vault length (Fig. 5). Occipitally deformed specimens displayed a most unusual pattern in that most had the posterior branch course straight and then angled 90° upwards as it neared the occipital bone (Figs. 7, 8). As with the anterior branch of the middle meningeal vessels, those skulls that were both craniosynostotic and artificially deformed had branches that followed the direction of altered cranial growth (Fig. 6).

DISCUSSION

Effects of craniosynostosis

Further information from clinical research is necessary to interpret the above results. Most individuals with craniosynostosis do not have reduced intracranial volumes; rather, their volumes fall within the normal range of variability. (Gault et al., 1990). However, in vivo studies have shown that many individuals do experience elevated intracranial pressure (Fok et al., 1992;

TABLE 5. Frequency of enlarged occipital/marginal sinuses

Group[1]	Presence		Asymmetry: Wider/ deeper O/M pattern on:		
	Yes	No	Left	Equal	Right
Undeformed	9	13	2	1	6
Total craniosynostoses	3	6	0	2	1
Sagittal synostosis only	2	3	0	1	1
Squamosal synostosis only	1	1	0	1	0
Sagittal + other synostoses	0	2			
Total occipital deformation	12	5	5	3	4
Left asymmetrical	4	2	2	1	1
No asymmetry	3	2	2	1	0
Right asymmetrical	5	1	1	1	3
Craniosynostosis and cranial deformation	0	2			

[1] Subgroups are in italics.

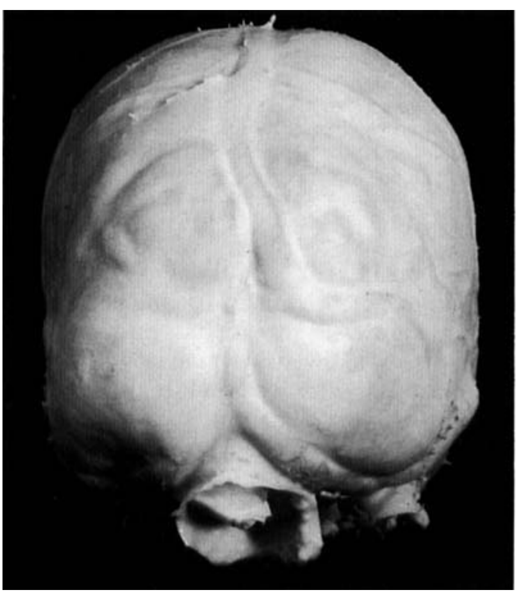


Fig. 4. Posterior view of an undeformed endocast with enlarged occipital and marginal sinuses. These sinuses are approximately the same width and depth as the transverse sinuses.

Gault et al., 1992). Thus, compared to normal individuals, many craniosynostotic subjects have approximately the same amount of endocranial space, but their endocranial contents are under a greater generalized intracranial pressure. Another important clinical finding is that cerebrospinal fluid (CSF) forms localized accumulations in areas of compensatory skull growth in young children (Chadduck et al., 1992). These accumulations represent relocation of CSF away from the synostotic areas and toward areas with unrestricted growth. CSF accumulations are greatly reduced or disappear once brain and skull growth are nearly complete (Chadduck et al., 1992).

In light of these findings, one may interpret the results of this research as showing that both increased intracranial pressure and localized enlarged areas of CSF directly affect other endocranial structures such as the venous sinuses. Venous sinuses are responsible for draining deoxygenated blood and CSF from the brain (Gray, 1973). An increase in pressure and/or an increase in CSF could hamper this drainage. Sinuses near areas of compensatory skull growth will not be restricted in their development, so these sinuses may not necessarily produce wider and deeper grooves in the bone. Furthermore, accumulations of CSF in areas of compensatory skull growth (Chadduck et al., 1992) probably provide greater than normal room for these venous sinuses to develop, since this CSF serves as a large cushion between the sinus and the brain. Sinuses can develop into the CSF accumulations rather than impinging upon the bone and making wider, deeper grooves. However, sinuses directly along or near a craniosynostotic suture are restricted in their displacement. In addition to bone growth restriction from suture closure, clinical evidence suggests that there is comparatively less CSF underneath the synostotic area (Chadduck et al., 1992), which indicates less than normal room for venous sinuses in this area to develop. The end result is that these restricted sinuses

TABLE 6. *Middle meningeal vessel patterns*[1]

Group[2]	Anterior branch										Posterior branch							
	Width				Depth				Convoluted		Width			Depth			Convoluted	
	S	M	L	XL	Shallow	Medium	Deep	Very deep	No	Yes	S	M	L	Shallow	Medium	Deep	No	Yes
Undeformed	5	23	15	1	3	26	13	2	29	15	15	28	1	16	27	1	43	1
Total craniosynostoses	0	10	7	0	0	11	6	0	7	10	2	15	0	1	14	2	15	2
Sagittal synostosis only	0	6	3	0	0	6	3	0	4	5	1	8	0	1	6	2	9	0
Squamosal synostosis only	0	1	3	0	0	2	2	0	2	2	0	4	0	0	4	2	4	0
Sagittal + other synostoses	0	3	1	0	0	3	1	0	1	3	1	3	0	0	4	0	2	2
Total occipital deformation	4	14	12	4	0	12	20	2	18	16	8	21	5	5	24	5	32	2
Left asymmetrical	2	6	3	1	0	4	7	1	3	9	2	8	2	1	10	1	12	0
No asymmetry	1	3	6	0	0	3	7	0	7	3	3	7	0	1	9	0	10	0
Right asymmetrical	1	5	3	3	0	5	6	1	8	4	3	6	3	3	5	4	10	2
Craniosynostosis and cranial deformation	0	0	4	0	0	1	3	0	0	4	1	3	0	0	4	0	2	2

[1] Values for right and left meningeal vessels are grouped together.
[2] Subgroups are in italics.

impinge on the bone, producing very wide, deep grooves.

Intuitively, one might assume a common endocranial characteristic and compensatory mechanism of sagittal synostosis is a very wide, deep impression for the superior sagittal sinus (Fig. 2). This research and the findings of others (Eiseley and Asling, 1944) support this hypothesis. Transverse and sigmoid sinuses did not display these same characteristics hypothetically because their development was not directly hampered, but rather facilitated by both compensatory skull growth and the postulated accumulations of CSF in the anterior-posterior dimension. In addition, transverse and sigmoid sinus blood flow could be relieved through the occipital/marginal sinuses. Skulls with squamosal synostosis lacked these dramatic changes because the area of synostosis did not directly overlie a major venous sinus. The specimen with both sagittal and lambdoidal synostosis provided further support for this hypothesis. Ortner and Putschar (1985) have previously presented this case as an example of enlarged parietal foramina. These sinuses were in close proximity to the areas of restricted growth. Since venous drainage may have been impeded, some blood flow was redirected through emissary veins found within the enlarged parietal foramina.

Not all specimens exhibited wide, deep sinuses in the area directly underneath the craniosynostosis. Variation in sinus width and depth has been noted by others (Hohenthal and Brooks, 1960) and may be caused by 1) genetic predisposition for or against wide, deep sinuses, 2) the timing of incidence of craniosynostosis, 3) the age of the individual (there are age-related differences in the amount of detail found on an endocranium), 4) the presence and duration of enlarged subarachnoid accumulations of CSF, and 5) the presence or absence of increased intracranial pressure (ICP) and/or other unknown factors. Although increased ICP is very common with craniosynostosis (Fok et al., 1992; Gault et al., 1992), there are numerous craniosynostotic individuals with normal ICP.

Other compensatory mechanisms may include the presence of enlarged occipital/marginal sinuses. Only three of the nine cranio-

TABLE 7. Middle meningeal vessel directional and branch patterns

Group	Anterior branch patterns	Posterior branch patterns
Undeformed	Vessel tends to be relatively straight and runs parallel to the coronal suture; number of terminal branches ranges from few to many, and their direction is varied	Vessel is very straight; it courses posteriorly to either the occipital region or toward lambda
Total craniosynostoses	Vessel tends to be slightly convoluted and runs parallel to the coronal suture	Characterized by a very long, straight vessel that follows a direction similar to that seen among the undeformed crania
Sagittal synostosis only	Vessel is slightly or greatly convoluted and runs parallel to the coronal suture; there are multiple terminal branches, and these branches slant superior-posteriorly	See description for total craniosynostoses
Squamosal synostosis	Vessel tends to be slightly convoluted and runs parallel to the coronal suture	See description for total craniosynostoses
Sagittal and other synostoses	Vessel tends to be slightly convoluted and runs parallel to the coronal suture	See description for total craniosynostoses
Craniosynostoses and artificial deformation	Vessel is slightly convoluted and runs parallel to the coronal suture; there are multiple terminal branches, and these branches slant superior-posteriorly, or in the direction of altered cranial growth	Vessel is straight; the course of the vessel and its branches follows the direction of altered cranial growth
Occipital deformation	Vessel usually is straight and runs parallel to the coronal suture; number of terminal branches varies, and frequently some branches are found in the area of the frontal lobe	Vessel is relatively straight; vessel courses posteriorly and then angles 90° upward as it nears the occipital bone

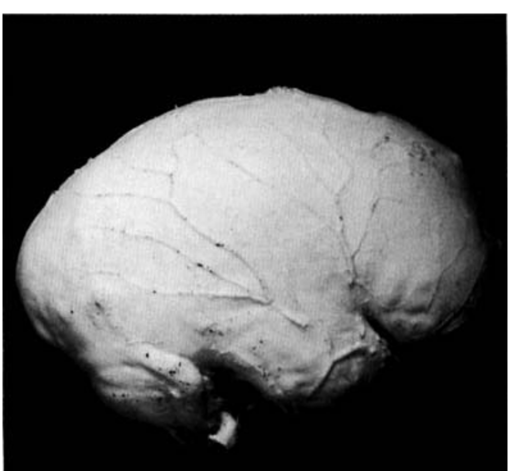


Fig. 5. Right view of an endocast with sagittal synostosis. Middle meningeal vessel impressions are deep and course superior-posteriorly, or in the direction of altered cranial growth.

Fig. 6. Left view of a skull exhibiting both parallelo-fronto-occipital deformation and sagittal synostosis and its corresponding endocast. Note the middle meningeal vessel directional patterns parallel the direction of altered cranial growth.

synostotic individuals displayed this trait, suggesting it is also influenced by other factors. Study of a larger craniosynostotic sample may determine if enlarged occipital/marginal sinuses are common.

Moss (1959) noted that craniosynostosis will redirect the growth vectors of the endocranial components. These patterns are clearly evident in the anterior and posterior branches of the middle meningeal vessels. Width and depth did not change dramatically, although the vessels were more convoluted. In response to the initial restriction and compensatory redirection of growth (and

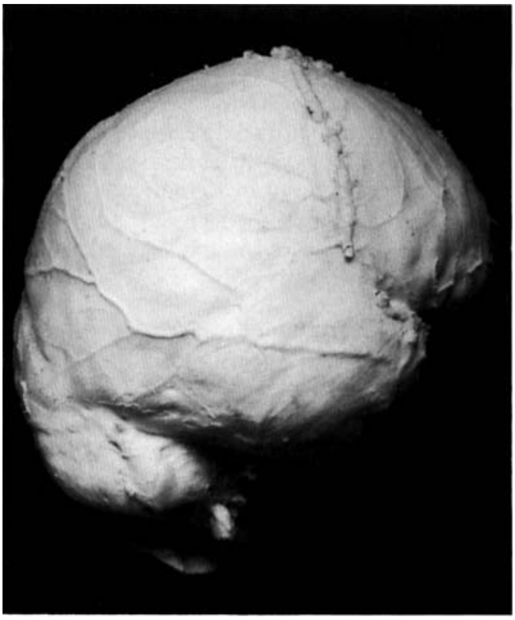


Fig. 7. Right view of a right asymmetrically deformed endocast. The anterior meningeal branch is very wide and deep, and the posterior branch angles 90° upward.

Fig. 8. Comparison of craniosynostotic **(left)** and occipitally deformed **(right)** endocasts. Although the endocranial shape for each is different, they are similar in that middle meningeal vessel patterns have been altered in similarly predictable patterns due to deformational constraints.

in addition to possible increased intracranial pressure), the vessels may become wider and make deeper impressions in the bone (Figs. 5 and 8). Both the anterior and posterior branches showed an increase in length, which demonstrates the developmental plasticity of the vessels as they adapted to the altered cranial growth. This plasticity also was seen in the sagittal synostotic individuals, whose anterior branches were slanted superior-posteriorly (i.e., in the direction of increased cranial length).

Effects of occipital deformation

Asymmetrical occipital deformation may affect superior sagittal sinus drainage (Dean, 1993, 1995a). This deformation directly compresses one of the transverse sinuses. In response, blood may be rerouted through the confluens sinuum, and the contralateral transverse sinus must accommodate increased blood flow demands. Supporting this was that left asymmetrically deformed endocasts generally had wider and deeper right transverse sinuses, whereas the reverse was true for right asymmetrically deformed endocasts (Fig. 3).

A word of caution, however, is warranted. In undeformed individuals, the predominant pattern of sagittal sinus deviation is toward the right transverse sinus, with only a small collateral channel connecting to the left transverse sinus (Knott, 1882; Gibbs and Gibbs, 1934; Browning, 1953; Gray, 1973; Hochberg and LeMay, 1974; Durgun et al., 1993). This drainage pattern is established in early prenatal life (Streeter, 1915, 1918; Padget, 1957). Thus, it cannot be determined indisputably from this sample whether the wider, deeper right transverse sinus in left asymmetrically deformed crania resulted from deformational processes or from an established prenatal pattern.

Asymmetrical deformation also appears to produce asymmetry in transverse and sigmoid sinus impressions. The right vessels tended to be wider and deeper among left asymmetrically deformed crania; however, left vessels were wider but not deeper among right asymmetrically deformed crania. A possible explanation for this is as follows. As one of the sinuses becomes obstructed or compressed, compensatory blood drainage occurs in the contralateral sinus. This increased blood flow is probably first expressed by a widening of the sinus. If the vessel is already wide and/or dominant (such as the right transverse sinus, which usually re-

ceives the majority of blood flow from the sagittal sinus), further width expansion probably is prevented by the limited space in the endocranium. Thus, the next step in accommodating a redirection of blood flow is for the already widened vessel to make a deeper groove in the bone.

Enlarged occipital/marginal (O/M) sinuses were displayed in 71% of the artificially deformed sample. Occasional enlargement of these sinuses has been recorded (Knott, 1882), yet the frequency for this trait is much lower than the 71% documented here (Gray, 1973; Falk, 1986). The undeformed population also displayed higher than average frequencies for these sinuses. One possibility is that endocasts provide greater detail of the endocranium vs. visual inspection of the skull. Thus, enlarged occipital/marginal sinuses may be overlooked if an endocast is not prepared. Further, my method of scoring may differ from that of other researchers, who may have more discrete methods for classifying O/M impressions or who may code for enlarged O/M sinuses in association with reduced transverse sigmoid sinuses (i.e., Tobias, 1967).

Despite the above concerns, the occipitally deformed group still has a much higher incidence of enlarged occipital/marginal sinuses compared to the undeformed group. Occipital deformation greatly affects the transverse and sigmoid sinuses. If blood flow is obstructed through these pathways, blood may be redirected through the adjacent occipital/marginal sinuses. Increased blood flow may cause enlargement of these vessels (Dean, 1993, 1995a, 1995b). Asymmetrical deformation also appears to affect both the presence and the asymmetry of this trait, since the wider and deeper occipital/marginal sinus system was found on the same side as the greatest deformational stress.

Developmental plasticity of the endocranial vasculature also was displayed in the middle meningeal vessel structure (Figs. 7, 8). The posteriorly placed deformation could obstruct posterior meningeal vessel flow. Consequently, the anterior branches have to compensate for this redirection of flow, resulting in relatively wider, deeper, more convoluted anterior vessels. Occipitally deformed endocasts also displayed an increased number of terminal branches on the frontal lobe, which suggest increased vascularization in those areas furthest from deformational effects. Redirection of the posterior branches was probably a direct effect of compression upon the occipital area. As posterior growth of these vessels was restricted by the occipital compression, vessel direction angled toward the path of least resistance, or superiorly.

Effects of craniosynostosis and artificial cranial deformation

Individuals with both craniosynostosis and artificial cranial deformation present an interesting research dilemma. Was the craniosynostosis the primary cause of growth alterations which were compounded by the artificial deformation, or was the craniosynostosis a secondary response to the artificial deformation? Differences between the effects of primary and secondary craniosynostosis have been noted in the clinical literature (Cohen, 1986a, 1988; Bixler and Ward, 1987; Bruneteau and Mulliken, 1992); however, distinguishing between the two in skeletons is extremely difficult.

Examining the endocranial alterations from the skulls with both craniosynostosis and artificial deformation can yield some insight (Fig. 6). Direction of the middle meningeal vessels followed that of the altered cranial growth, a finding that was similar in both craniosynostotic crania and occipitally deformed crania. However, sagittal sinus width and depth resembled that of the artificially deformed group, rather than the craniosynostotic group. This finding suggests that for the specimens used in this research, sagittal synostosis was a consequence of the artificial cranial deformation.

Comparisons between craniosynostosis and occipital deformation

Craniosynostosis and positional deformation, although grossly morphologically similar, demonstrate subtle ectocranial morphological differences (Bruneteau and Mulliken, 1992). A similar pattern could arise endocranially, For example, positionally deformed children do not display the localized accumulation patterns of CSF that are char-

acteristic of craniosynostotic children (Chadduck et al., 1992).

This research showed venous sinus responses to either craniosynostosis or artificial cranial deformation differ. Perhaps due to elevated intracranial pressure (Fok et al., 1992; Gault et al., 1992) and/or localized accumulations of CSF away from the synostotic area (Chadduck et al.,1992), the sinuses directly underlying synostosed sutures were restricted in their movement and thus made wider and deeper impressions on the bone. Localized accumulations of CSF and increased intracranial pressure are not to be expected in normal individuals whose skulls were artificially deformed (Chadduck et al., 1992). In this study, venous sinuses under the greatest deformational pressure were compressed, and there was compensatory enlargement of the sinuses responsible for handling the increase in blood flow. However, middle meningeal vessel patterns were quite similar for both groups (Fig. 8). The direction of these vessels followed the altered direction of cranial growth, in response to the need for vascularization in specific endocranial areas.

Enlarged occipital/marginal sinuses

The high incidence of enlarged occipital/marginal sinuses among three of the four groups deserves further discussion (see Figs. 3, 4). These results and earlier findings (Dean, 1993, 1995a, 1995b) suggest that different kinds of cranial deformation influence the size and shape of the sinuses. Thus, these sinuses, along with the rest of the endocranial vasculature, appear to be developmentally plastic. Should there be factors that affect this plasticity, such as cranial deformation, the end result can be great variability between the vasculature of undeformed and deformed specimens.

The plasticity of the occipital/marginal sinuses merits further study, since enlarged occipital/marginal sinuses have been used as a synapomorphic trait for reconstructing hominid phylogenies (Falk and Conroy, 1983; Falk, 1986, 1988; Conroy et al., 1990; Tobias and Symons, 1992). My findings suggest this trait may not be as conservative as previously thought and may be highly susceptible to pathological or cultural influences. Thus, these influences need to be considered when assessing the phylogenetic implications of enlarged occipital/marginal sinuses.

CONCLUSIONS

This research has shown that both artificial cranial deformation and craniosynostoses dramatically alter endocranial features. Altered middle meningeal vessel patterns were similar between the two groups, but venous sinus patterns varied. I propose that increased intracranial pressure and localized accumulations of CSF in areas of compensatory skull growth are responsible for the venous sinus changes found in craniosynostotic individuals. Further research is needed to test this hypothesis.

ACKNOWLEDGMENTS

Preliminary findings were presented at the 1994 American Association of Physical Anthropology meetings (Dean, 1994). I appreciate the efforts of Dr. Della Cook and Dr. Kevin Hunt in their critical reviews of this manuscript. I also thank the anonymous reviewers for their comments regarding this manuscript. The crania used in this study were provided by Indiana University, the Thomas Gilcrease Institute of American History and Art (Tulsa, OK), and the National Museum of Natural History, Smithsonian Institution (Washington, DC). I thank these institutions for granting me access to the skeletal collections and assisting me wherever possible. I am grateful to members of the Anthropology Conservation staff at the Smithsonian Institution for their suggestions and for providing me workspace in their lab. Exhibits Specialist Paul Rhymer at the Office of Exhibits Central was instrumental in providing information about endocast materials and preparation. Finally, I am indebted to Dr. David Hunt, Physical Anthropology Collections Manager at the Smithsonian Institution, for not only granting me access to the skeletal collections but for his assistance with all aspects of my research throughout my 7 month stay. His help, comments, and guidance were greatly appreciated.

LITERATURE CITED

Anton SC (1989) Intentional cranial vault deformation and induced changes of the cranial base and face. Am. J. Phys. Anthropol. *79:*253–267.

Bennett K (1967) Craniostenosis: A review of the etiology and a report of new cases. Am. J. Phys. Anthropol. *27:*1–10.

Bixler D, and Ward R (1987) Craniosynostosis. In N Myrianthopoulos (ed.): Handbook of Clinical Neurology, Vol. 6(50). pp. 113–128.

Bjork A, and Bjork L (1964) Artificial deformation and cranio-facial asymmetry in ancient Peruvians. J. Dent Res. *43:*353–362.

Browning H (1953) The confluence of dural venous sinuses. Am. J. Anat. *93:*307–329.

Bruneteau RJ, and Mulliken JB (1992) Frontal Plagiocephaly: Synostostic, Compensational, or Deformational. Plast. Reconstr. Surg. *89:*21–31.

Chadduck WM, Chadduck JB, and Boop FA (1992) The subarachnoid spaces in craniosynostosis. Neurosurgery *30:*867–871.

Cheverud JM, and Midkiff JE (1992) Effects of fronto-occipital cranial reshaping on mandibular form. Am. J. Phys. Anthropol. *87:*167–172.

Cheverud JM, Kohn LAP, Konigsberg LW, and Leigh SR (1992) Effects on fronto-occipital artificial cranial vault modification on the cranial base and face. Am. J. Phys. Anthropol. *88:*323–346.

Cohen MM (1986a) History, terminology and classification of craniosynostosis. In: Craniosynostosis: Diagnosis, Evaluation and Management. New York: Raven Press, pp. 1–20.

Cohen MM (1986b) The etiology of craniosynostosis. In: Craniosynostosis: Diagnosis, Evaluation and Management. New York: Raven Press, pp 59–79.

Cohen MM (1986c) Perspectives on craniosynostosis. In: Craniosynostosis: Diagnosis, Evaluation and Management. New York: Raven Press, pp. 21–57.

Cohen MM (1988) Craniosynostosis update 1987. Am. J. Med. Genet. Suppl. *4:*99–148.

Conroy G, Vannier M, and Tobias PV (1990) Endocranial features of *Australopithecus africanus* revealed by 2- and 3-D computed tomography. Science, *247:*838–841.

Dean VL (1993) Induced changes in sinus and meningeal vessel patterns due to artificial cranial deformation. Am. J. Phys. Anthropol. *(Suppl. 16):*79 (abstract).

Dean VL (1994) Comparative endocranial effects of craniosynostosis and artificial cranial deformation. Am. J. Phys. Anthropol. *(Suppl 18):*77 (abstract).

Dean VL (1995a) Sinus and meningeal vessel pattern changes induced by artificial cranial deformation. Int. J. Osteoarchaeol. *5:*1–14.

Dean VL (1995b) Effects of cultural cranial deformation and craniosynostosis on cranial venous sinus and middle meningeal vessel patterns expression. Ph.D. thesis, Indiana University, Bloomington, IN.

Delashaw JB, Persing JA, Broaddus WC, and Jane JA (1989) Cranial vault growth in craniosynostosis. J. Neurosurg. *70:*159–165.

Diamond MK (1991) Homologies of the stapedial artery in humans, with a reconstruction of the primitive stapedial artery configuration of euprimates. Am. J. Phys. Anthropol. *84:*433–462.

Diamond MK (1992) Homology and evolution of the orbitotemporal venous sinuses. Am. J. Phys. Anthropol. *88:*211–244.

Dingwall EJ (1931) Artificial Cranial Deformation: A Contribution to the Study of Ethnic Mutilations. London: John Bale, Sons and Danielsson, Ltd.

Durgun B, Ilgit ET, Cizmeli MO, and Atasever A (1993) Evaluation by angiography of the lateral dominance of the drainage of the dural venous sinuses. Surg. Radiol. Anat. *15:*125–130.

Eiseley L, and Asling CW (1944) An extreme case of scaphocephaly from a mound burial near Troy, Kansas. Trans. Kan. Acad. Sci. *47:*241–255.

Falk D (1986) Evolution of cranial blood drainage in hominids: Enlarged occipital/marginal sinuses and emissary foramina. Am. J. Phys. Anthropol. *70:* 311–324.

Falk D (1988) Enlarged occipital/marginal sinuses and emissary foramina: Their significance in hominid evolution. In FE Grine (ed.): Evolutionary History of the "Robust" Australopithecines. New York: Aldine de Gruyter, pp. 85–96.

Falk D (1993) Meningeal arterial patterns in great apes: Implications for hominid vascular evolution. Am. J. Phys. Anthropol. *92:*81–97.

Falk D, and Conroy G (1983) The cranial venous sinus system in *Australopithecus afarensis.* Nature *306:* 779–781.

Falk D, and Nicholls P (1992) Meningeal arteries in rhesus macaques (Macaca mulatta): Implications for vascular evolution in anthropoids. Am. J. Phys. Anthropol. *89:*299–308.

Fok H, Jones BM, Gault DG, Andar U, and Hayward R (1992) Relationship between intracranial pressure and intracranial volume in craniosynostosis. Br. J. Plast. Surg. *45:*394–397.

French LR, Jackson IT, and Melton LJ (1990) A population-based study of craniosynostosis. J. Clin. Epidemiol. *43:*69–73.

Gault DT, Renier D, Marchac D, Ackland FM, and Jones BM (1990) Intracranial volume in children with craniosynostosis. J. Craniofac. Surg. *1:*1–3.

Gault DT, Renier D, Marchac D, and Jones BM (1992) Intracranial pressure and intracranial volume in children with craniosynostosis. Plast Reconstr. Surg. *90:*377–381.

Gibbs EL, and Gibbs FA (1934) The cross section areas of the vessels that form the torcular and the manner in which flow is distributed to the right and left lateral sinus. Anat. Rec. *59:*419–426.

Giuffrida-Ruggeri V (1912) Uber die Endocranischen Furchen der Arteria Meningea Media Beim Menschen. Z. Morphol. Anthropol. *15:*401–415.

Gray H (1973) Gray's Anatomy. Philadelphia: W.B. Saunders Co. (35th British edition).

Grupe G (1984) On diploic structures and their variability in artificially deformed skulls. J. Hum. Evol. *13:*307–309.

Hauser G, and Kritscher H (1994) Brief communication: The skull attributed to Mozart—craniosynostosis or not? Am. J. Phys. Anthropol. *94:*263–268.

Hochberg FH, and LeMay M (1974) Arteriographic correlates of handedness. Neurology. *25:*218–222.

Hohenthal WD, and Brooks ST (1960) An archaeological

scaphocephal from California. Am. J. Phys. Anthropol. *18*:59–67.

Holliday DY (1993) Occipital lesions: A possible cost of cradleboards. Am. J. Phys. Anthropol. *90*:283–290.

Hrdlicka A (1910) Artificial deformations of the human skull, with especial reference to America. Int. Cong. Americanists: Actas. *17*:147–149.

Knott JF (1882) On the cerebral sinuses and their variations. J. Anat. *16*:27–42.

Kohn LAP, Leigh SR, Jacobs SC, and Cheverud JM (1993) Effects of annular cranial vault modification on the cranial base and face. Am. J. Phys. Anthropol. *90*:147–168.

Lindblom K (1936) Roentgenographic study of the vascular channels of the skull. Acta Radiol. *(Suppl. 30)*: 1–146.

Moss ML (1958) The pathogenesis of artificial cranial deformation. Am. J. Phys. Anthropol. *16*:269–286.

Moss ML (1959) The pathogenesis of premature cranial synostosis in man. Acta Anat. *37*:251–370.

Moss ML, and Young RW (1960) A functional approach to craniology. Am. J. Phys. Anthropol. *18*:281–292.

Murrill RI, and Wallace DT (1971) A method for making an endocranial cast through the foramen magnum of an intact skull. Am. J. Phys. Anthropol. *34*:441–446.

Neumann GK (1942) Types of artificial cranial deformation in the eastern United States. Am. Antiquity *3*:306–318.

Oetteking B (1927) Pathologic plagiocephaly in a Nevada skull. Indian Notes *4*:201–209.

Ortner DJ, and Putschar WGJ (1985) Identification of Pathological Conditions in Human Skeletal Remains. Washington, DC: Smithsonian Institution Press.

Ossenberg NS (1970) The influence of artificial cranial deformation on discontinuous morphological traits. Am J. Phys. Anthropol. *33*:357–372.

Padget DH (1957) The development of the cranial venous system in man, from the viewpoint of comparative anatomy. Contrib. Embryol. *36*:81–151.

Persing JA, and Jane JA (1989) Craniosynostosis. Semin. Neurol. *9*:200–209.

Persing JA, Jane JA, and Shaffrey M (1989) Virchow and the pathogenesis of craniosynostosis: A translation of his original work. Plast. Reconstr. Surg. *83*:738–742.

Radinsky LB (1968) A new approach to mammalian cranial analysis; illustrated by examples of prosimian primates. J. Morphol. *124*:167–180.

Rothman D (1937) The endocranial course of the middle meningeal artery in American whites and American Negroes. Am. J. Phys. Anthropol. *22*:425–435.

Saunders JB, and O'Malley CD (1950) The Illustrations from the Works of Andreas Vesalius of Brussels, With Annotations and Translations, a Discussion of the Plates and Their Background, Authorship and Influence, and a Biographical Sketch of Vesalius. New York: Dover Publications.

Schendel SA, Walker G, and Kamisugi A (1980) Hawaiian craniofacial morphometrics: Average Mokapuan skull, artificial cranial deformation, and the "rocker" mandible. Am. J. Phys. Anthropol. *52*:491–500.

Streeter GL (1915) The development of the venous sinuses of the dura mater in the human embryo. Am. J. Anat. *18*:145–178.

Streeter GL (1918) The developmental alterations in the vascular system of the brain of the human embryo. Contrib. Embryol. *8*:5–38.

Tobias PV (1967) The Cranium and Maxillary Dentition of *Australopithecus (Zinjanthropus) boisei*. Olduvai Gorge, Vol. 2. Cambridge: Cambridge University Press.

Tobias PV, and Symons JA (1992) Functional, morphogenetic and phylogenetic significance of conjunction between cardioid foramen magnum and enlarged occipital and marginal venous sinuses. Persp. Hum. Biol. 2/Archaeol. Oceania *27*:143–152.

Wood Jones F (1912) On the grooves upon the ossa parietalia commonly said to be caused by the arteria meningea media. J. Anat. Phys. *46*:228–238.